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Estradiol, estrogen receptor alpha, and osteogenic responses to mechanical loading
Maureen J. Devlin, Daniel E. Lieberman, Bjorn R. Olsen, Naomi Fukai

Abstract:
Despite evidence that mechanical loads can induce diaphyseal bone growth, there is little consensus about how, and to what extent, strain affects human skeletal phenotype. This project tests a mechanism of mechanotransduction in bone that may underlie variation in human skeletal robusticity. One hypothesis of particular relevance to humans is that hormones, particularly estradiol (E_2) and its receptor, estrogen receptor alpha (ER-a), affect mechanotransduction in osteoblasts. Previous experiments demonstrate that E_2 increases osteogenic responses to loading, but the mechanism involved is unclear. This project tests the hypothesis that E_2 affects osteogenesis by upregulating ER-a, making osteoblasts more sensitive to mechanical loading.
To test this hypothesis, 36 ovarie c-tomized C57BL/6J mice were divided into normal, high, and low E_2 treatment groups implanted with 0.25 mg, 2.5 mg, or placebo E_2 pellets. Half of the mice in each E_2 group were fed normal mouse pellets, while half were fed a soft paste made from the same pellets. Results indicate that in the lateral mandibular corpus, hard diet animals exhibit 260% (high E_2), 21% (low E_2), and 82% (placebo) more growth than soft diet animals ($p=0.01$ to 0.03). In situ hybridization shows that ER-a is expressed in mandibular osteoblasts and hypertrophic chondrocytes, and expression appears to increase with increasing E_2 dose. Evidence that E_2 level affects diaphyseal bone growth via regulation of ER-a may help explain patterns of variation in human skeletal robusticity.